

Investigating Age Differences in the Genetic and Environmental Structure of the Tridimensional Personality Questionnaire in Later Adulthood

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In this study we examined cross-sectional age differences in means, phenotypic covariance structure, and the underlying genetic and environmental structure of four personality constructs from Cloninger's personality system: Novelty Seeking (NS), Harm Avoidance (HA), Reward Dependence (RD), and Persistence (PS). Study participants were same-sex female twins between the ages of 50 and 96, drawn from the American Association of Retired Persons (AARP) twin sample. We examined age differences by comparing younger (age 50–65) and older (age 66+) cohorts (based on a median split of the sample) and by estimating biometrical model parameters as linear and quadratic functions of continuous age. Results indicated modest, but significant, mean-level declines across this age range for NS, RD, and PS. HA showed no significant mean differences. We found moderate heritability estimates for all of the TPQ higher-order personality dimensions, ranging from 0.16 to 0.62. No significant age differences in the proportion of genetic and environmental influences on the TPQ dimensions were found. For HA, RD, and PS there were no significant age-related differences in total variance. However, for NS we observed a decline in total phenotypic variance across age cohorts.

KEY WORDS: Personality; genetics; TPQ; aging; twins.

INTRODUCTION

An important issue in studying the life course development of personality is whether personality changes can be attributed to biological influences (e.g., genetic factors) or to social or environmental factors. Behavioral genetic analyses of personality traits at different stages of the life course can help to address this question by partitioning phenotypic trait variance into three sources: variance due to genetic factors, variance explained by environmental variables shared by members of the same family, and variance due to idiosyncratic, or individual, experience.

Although there is a considerable literature examining the development of personality constructs at varying times from childhood to adulthood (e.g., Goldsmith, 1983; McCartney, Harris, and Bernieri, 1990; Carmichael and McGue, 1994; Viken *et al.*, 1994), there is a relative paucity of research investigating age-related personality changes in later adulthood. The study of personality in the context of aging is important, since personality, as a set of characteristic dispositions, plays an important role in emotional, interpersonal, experiential, attitudinal, and motivational styles (Schroots, 1996). Later adulthood is a time characterized by substantial changes that can be both environmental (e.g., retirement, widowhood) and biological (e.g., decreasing physical potency, menopause). It is thus important to understand the etiology of personality stability and change in the face of such significant events in this particular time of the life span.

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There have been numerous theoretical developments in the psychology of aging in the last half of the past century. Regarding personality and aging, Schroots (1996) distinguishes between two theoretical traditions—trait and developmental-stage models. In both traditions, the central issue concerns the extent and nature of personality stability and change over the life course. Behavioral genetic studies of personality and aging are concerned with the extent to which environmental and hereditary factors influence these trait and developmental-stage changes over the life span. In general, studies of adult populations suggest that personality in adulthood is shaped primarily by genetic influences and unique environmental experience. There is very little evidence that shared familial influences affect personality development, particularly after adolescence (Eaves, Eysenck, and Martin, 1989; Loehlin, 1992; Loehlin, Horn, and Willerman, 1998). In terms of personality change, unique environmental influences appear to account for a substantial proportion of the variance in personality change across adulthood, while the stable core of personality appears to be associated with genetic factors (McGue, Bacon, and Lykken, 1993; Viken *et al.*, 1994; Pedersen and Reynolds, 1998). Furthermore, age differences in personality across the adult life span appear to be universal across cultures (McCrae *et al.*, 1999, 2000), suggesting an intrinsic developmental component to personality.

However, few studies have included sufficient numbers of older adults to determine whether these findings generalize to the later stages of the life course. And much of the available data concerns only the higher-order personality dimensions of Extraversion (E) and Neuroticism (N) from the Big Five or Eysenck's system (e.g., Pedersen *et al.*, 1988; Tambs *et al.*, 1991; Heath *et al.*, 1992; Bergeman *et al.*, 1993; Carmichael and McGue, 1994; Viken *et al.*, 1994; Jang, Livesley, and Verson, 1996; Loehlin, Horn, and Willerman, 1998; Eaves *et al.*, 1999). We are not aware of any studies examining Cloninger's (1986) personality dimensions in older adults. Cloninger developed a neurobiological-based theory of personality that attempts to measure genetically homogeneous and independent temperament dimensions, while most personality traits have been characterized on the basis of factor analyses of the phenotypic structure of personality. Although Cloninger's dimensions have been shown to correlate with other personality constructs (e.g., Heath, Cloninger, and Martin, 1994; Stallings *et al.*, 1996), Heath *et al.* (1994) showed that the personality systems

of Eysenck and Cloninger are not alternative descriptions of the same dimensions of personality.

The purpose of the present study was to examine genetic and environmental influences on the higher-order personality dimensions assessed by Cloninger's Tridimensional Personality Questionnaire (TPQ; Cloninger, 1987) in women over age 50. Consistent with Cloninger's revised scoring (Cloninger, Svrakic, and Przybeck, 1993), we scored the TPQ to measure four personality or temperament dimensions: Harm-Avoidance (HA), Novelty-Seeking (NS), Reward-Dependence (RD), and Persistence (PS). In the present study, we conducted two analyses. First, we investigated differences in means and the phenotypic variance-covariance structure of the four dimensions, across two age cohorts (50–65 and 66–89 years of age; based on a median split of the sample). Biometric genetic analyses were then used to investigate age differences in genetic and environmental influences on individual differences in the personality dimensions across the cohorts. Second, we used age as a continuous variable and estimated phenotypic means, and genetic and environmental influences as a continuous function of age across the full age range of the sample from 50 to 96 years of age.

METHOD

Participants

The sample was a subset of twins who responded voluntarily to a notice appearing in a national newsletter published by the American Association of Retired Persons (AARP) between 1985 and 1989. If either twin responded to this recruitment, both pair members were mailed an initial questionnaire that assessed general health and lifestyle. In late 1990 and early 1991, respondents to this initial questionnaire were mailed a follow-up questionnaire that included Cloninger's TPQ (Cloninger, 1987). Valid questionnaires were obtained from 4119 participants, representing a response rate of 65%. The full sample was 74% female (3049 women and 1070 men). Participants were Caucasian twins between 50 and 96 years of age in 1991. The sample was restricted to Caucasian twins by design (see Meyer, Heath, and Eaves, 1992). For this study we excluded men, since too few male DZ twins were ascertained for valid age-based comparisons (data were available for only 65 complete DZ male twin pairs).

Our analysis 1 sample consisted of 849 complete female twin pairs between the ages of 50 and 89 (note: singletons were as old as 96, but the oldest complete

pair was 89 years of age), with a median age of 65 (577 female MZ twin pairs, and 272 female DZ same-sex twin pairs; 439 twin pairs in the younger cohort, and 410 twin pairs in the older cohort). For analysis 2 all available data were utilized. The analysis 2 sample consisted of 2220 to 2237 females (849 complete pairs from analysis 1 + 522–539 single-responding twins from incomplete pairs) between the ages of 50 and 96. The total *N* varies slightly due to differential patterns of missing data for some of the TPQ dimensions.

Zygosity Judgments

Zygosity was determined through standard questionnaire procedures that assessed the degree of physical similarity among the twins. If both twins classified themselves as identical, and both stated that they were frequently or sometimes confused with one another, they were classified as monozygotic (MZ). If either twin classified themselves as fraternal or responded that they were rarely confused with one another, they were classified as dizygotic (DZ). This classification scheme was conservative, with misclassifications most likely to be MZ twins incorrectly assigned as DZ twin pairs. Thus any biasing effects would be in the direction of overestimating the similarity of DZ twins. For more details regarding zygosity assignments, see Prescott *et al.* (1994).

Measures

TPQ

The TPQ assumes the observed phenotypic structure reflects the underlying biogenetic structure of personality. It was designed to correspond more closely to the underlying genetic structure of personality than other instruments derived by factor analysis of self-reported behavior (Cloninger, Svrakic, and Przybeck, 1993). The version used in the present study was a 100-item self-administered true-false instrument (Cloninger, 1987). The TPQ was originally designed to assess 12 primary subscales, 4 for each of the three higher-order personality dimensions as defined by Cloninger's theory: NS, HA, and RD. However, consistent with Cloninger's revised model (Cloninger, Svrakic, and Przybeck, 1993), we scored RD as the sum score of only 3 subscales: sentimentality versus insensitiveness (RD1), attachment versus detachment (RD3), and dependence versus independence (RD4). We scored persistence versus irresoluteness (RD2) as a separate primary dimension. NS was scored as the sum score

over its respective subscales: exploratory excitability versus stoic rigidity (NS1), impulsiveness versus reflection (NS2), extravagance versus reserve (NS3), and disorderliness versus regimentation (NS4). HA was scored as the sum total over its 4 subscales: anticipatory worry versus uninhibited optimism (HA1), fear of uncertainty versus confidence (HA2), shyness with strangers versus gregariousness (HA3), and fatigability and asthenia versus vigor (HA4). Internal consistencies (Cronbach's alpha) for the higher-order TPQ dimensions were 0.88, 0.72, 0.69, and 0.58 for HA, NS, RD, and PS, respectively (Stallings *et al.*, 1996).

Examples of NS items include "I'm slow to get excited about new ideas" (reverse scored); "I think in detail before deciding" (reverse scored); and "I do things spontaneously." Examples of HA items include "I'm confident that things will go well" (reverse scored); "I get tense and worried in unfamiliar situations"; "I avoid meeting strangers"; and "I have less energy than most." The revised RD scale consisted of items such as "I'm strongly moved by sentimental appeals"; "I don't open up much even with friends" (reverse scored) and "Others think I am too independent" (reverse scored). Items characteristic of the PS scale included "I often push myself to exhaustion"; "I work long after others give up"; and "I am satisfied with my accomplishments and have little desire to do better" (reverse scored).

Analyses

Analysis 1

Phenotypic analyses investigated age differences in means and variances, as well as the covariance structure of the four dimensions by comparing the younger (age 50–65) and older (age 66–89) cohorts. This was done using standard χ^2 difference tests, comparing constrained phenotypic means and variance-covariance structures across cohorts to corresponding parameters estimated freely within each cohort.

Biometrical genetic analyses (see Neale and Cardon, 1992) were then utilized to investigate potential heterogeneity in genetic and environmental influences on individual differences in the personality dimensions across the cohorts. Because Cloninger's scales are theoretically independent of one another, univariate biometrical model-fitting analyses were performed on each scale separately. Univariate analyses were performed using the structural equation modeling program Mx (Neale, 1999). To investigate the relative importance of genetic and environmental factors in explaining variation

in the TPQ dimensions, a series of hierarchically nested models were examined. We hypothesized four latent sources of variation: additive genetic factors (A), shared environmental factors (C), nonadditive (dominant) genetic factors (D), and nonshared environmental factors (E). Parameter estimates for additive genetic influences (a), shared environmental influences (c), nonadditive (dominant) genetic influences (d), and nonshared environmental influences (e) were estimated for each TPQ dimension. Note, however, that with twins reared together, as in our sample, shared environmental influences, and nonadditive genetic influences are confounded and cannot be estimated simultaneously in a given age cohort (Neale and Cardon, 1992). For each analysis, we compared the results allowing different parameter estimates in younger and older cohorts (i.e., unconstrained models) to models constraining parameter estimates to equality across cohorts. All models were fit to four 2×2 (twin 1 by twin 2) variance-covariance matrices: MZ younger cohort, DZ younger cohort, MZ older cohort, and DZ older cohort. Standard χ^2 difference tests were used to evaluate the fit of alternative nested models (see Neale and Cardon [1992] for details). Non-nested models were compared using Akaike's information criterion (AIC; Akaike, 1974).

Analysis 2

In a second analysis we used age as a continuous variable and estimated phenotypic means and genetic and environmental influences as a function of age and age-squared across the full age range from 50 to 96 years using Mx's raw data and definitional variable provisions. That is, all biometric model parameters were estimated as an intercept or average sample value, plus linear and quadratic terms as shown in equations 1 through 3 below. For the twin means

$$M = G + \beta_1 X + \beta_2 Y \quad (1)$$

where M equals the vector of expected twin means, G equals the grand mean or average sample value, X is age (i.e., using age as a definitional variable in Mx), Y is age squared (also a definitional variable), β_1 is the linear age difference parameter for the means, and β_2 is the quadratic age difference parameter. For the variance components

$$h^2 = (jj') + \beta_3 X + \beta_4 Y \quad (2)$$

where j is the path coefficient corresponding to the additive genetic latent factor, j' is the transpose of j, X and Y are age and age squared as defined above, β_3 is

the linear age difference parameter for the additive genetic effects, and β_4 is the quadratic age difference parameter. Environmental influences were estimated in a similar way:

$$e^2 = (kk') + \beta_5 X + \beta_6 Y. \quad (3)$$

Note that separate age difference parameters were estimated for the means and variance components parameters. For simplicity, only equations for additive genetic effects and nonshared environmental effects are shown, but other variance components (shared environmental effects and nonadditive genetic effects) were modeled in a similar fashion.

The effects of age on mean levels and variance components estimates were tested by sequentially setting the quadratic and linear terms to zero and comparing model fit to the full model. Again, standard χ^2 difference tests were used to evaluate alternative models. The advantage of this analysis is that it avoids arbitrary cutoffs to define age groups and also allows for investigating nonlinear age/time of measurement trends that would be difficult to detect in group comparison analyses.

RESULTS

Analysis 1

In order to allow our age cohort analyses to be comparable to the continuous age analyses where the effects of age are specifically modeled, we age-corrected our analysis 1 sample scores using standard regression procedures (i.e., we obtained residual scores after predicting each personality dimension from age and age squared). The age-corrected twin correlations and sample sizes are shown in Table I.

For the younger cohort note that the MZ correlation for NS is only slightly higher than the correlation for DZ twins, suggesting shared environmental effects for NS in the younger cohort. In the older cohort, however, the MZ correlation is almost four times the DZ correlation, suggesting dominance effects. For HA the opposite trend is apparent: dominance effects in the younger cohort and shared environmental effects in the older cohort. MZ correlations for RD and PS are about twice the DZ correlations in both age cohorts, suggesting additive genetic effects.

Cohort means and variances for each of the personality dimensions are shown in Figs. 1a and 2a, respectively. Results of cohort mean comparisons (χ^2 difference tests) indicated modest but significant declines in mean levels for RD, NS, and PS ($\Delta\chi^2_{(1)} = 16.0$,

Table I. Age-Corrected Correlations for TPQ Scales

Ages 50–65									
Young MZ female twins (N = 308)					Young DZ female twins (N = 131)				
	NS	RD	HA	PS		NS	RD	HA	PS
NS	0.42	0.17	−0.25	−0.03	NS	0.37	0.33	−0.21	0.04
RD	0.27	0.38	0.01	−0.02	RD	0.24	0.18	0.06	0.02
HA	−0.18	−0.09	0.52	0.07	HA	−0.09	0.01	0.10	−0.02
PS	0.01	0.08	−0.01	0.16	PS	−0.06	0.05	0.03	0.08
Ages 66–89									
Old MZ female twins (N = 269)					Old DZ female twins (N = 141)				
	NS	RD	HA	PS		NS	RD	HA	PS
NS	0.47	0.20	−0.20	−0.09	NS	0.12	0.38	−0.21	0.11
RD	0.24	0.35	−0.07	0.06	RD	0.21	0.17	−0.06	0.16
HA	−0.21	−0.15	0.46	0.04	HA	−0.06	−0.13	0.37	−0.08
PS	−0.08	0.15	−0.05	0.26	PS	−0.09	0.11	0.03	0.13

Note: Above the diagonal = twin 1 correlations; below the diagonal = twin 2 correlations; main diagonal = twin 1 – twin 2 correlations.

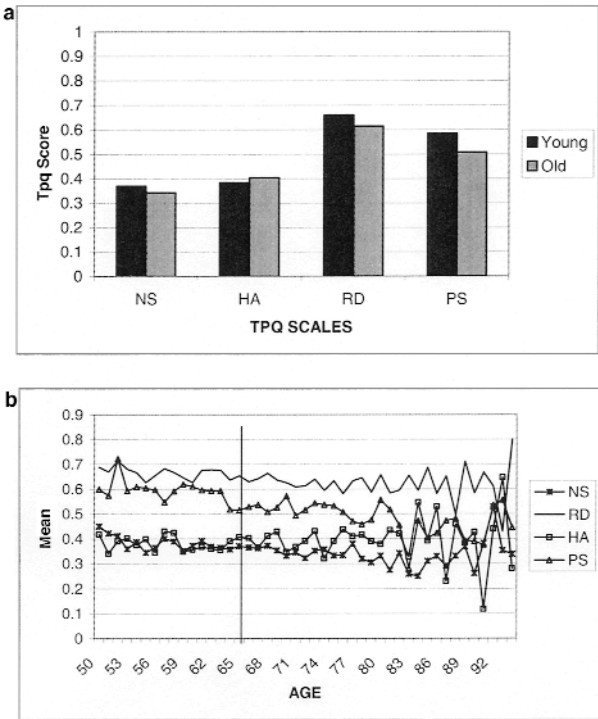


Fig. 1. (a) Observed means for the younger and older cohorts. (b) Observed means at each age. Note: the vertical line indicates the median split for analysis-1.

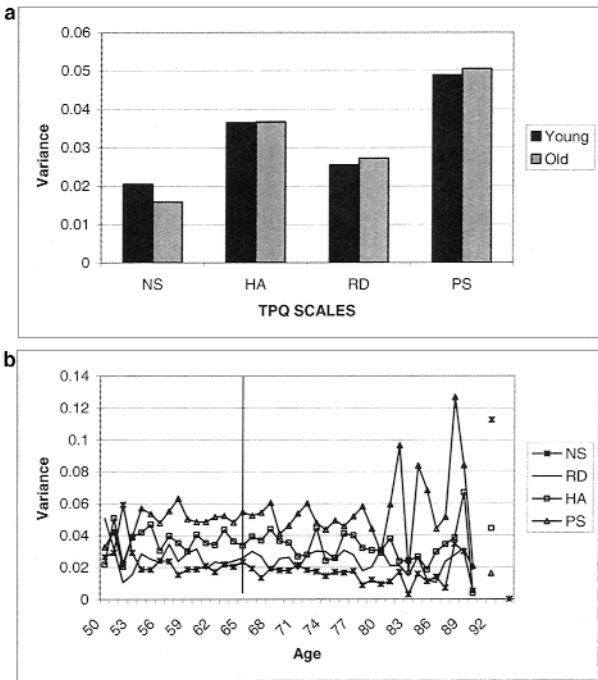


Fig. 2. (a) Observed variances for the younger and older cohorts. (b) Observed variances at each age. Note: the vertical line indicates the median split for analysis-1.

$p < .001$; $\Delta\chi^2_{(1)} = 30.6$, $p < .001$; $\Delta\chi^2_{(1)} = 54.0$, $p < .001$, respectively). No significant mean difference was found for HA. Phenotypic variances could be constrained to equality across age cohorts for all the personality dimensions except NS, which showed a 22% decline in the older cohort ($\Delta\chi^2_{(1)} = 7.63$, $p < 0.01$). We also found comparable covariances among the dimensions across the two cohorts, with the exception of the covariance between HA and RD ($\Delta\chi^2_{(1)} = 5.12$, $p < .05$) and between RD and PS ($\Delta\chi^2_{(1)} = 14.53$, $p < .001$). Although these covariances could not be constrained across cohorts due to our relatively large sample size, it should be noted that the absolute differences in the correlations were modest ($r = .01$ and $-.10$ between HA and RD in the younger and older cohorts, respectively; and $r = .01$ and $.15$ between PS and RD in the younger and older cohorts, respectively). These results suggest substantial stability in the phenotypic variances and covariances (i.e., structural stability) of the TPQ dimensions across cohorts.

Our observed means were very consistent with Cloninger's U.S. normative data (Cloninger, Przybeck, and Svrakic, 1991). Cloninger's means (reported as sum totals across items—but divided by the total number of items in each scale for presentation here) for adult white females, between the ages of 18 and 88 with a mean age of 45.3, were 0.38 for NS, 0.38 for HA, and 0.67 for RD. Our sample means were 0.37 and 0.34 for NS, 0.38 and 0.40 for HA, and 0.66 and 0.61 for RD, for the younger and older cohorts, respectively. Note that Cloninger's RD data and ours cannot be directly compared because, consistent with his earlier scoring, his normative RD scores include the PS scale, which we scored as a separate dimension.

Table II shows univariate model-fitting results for the four TPQ scales. We were able to drop shared environmental influences (c^2) and nonadditive (dominant) genetic influences (d^2) without significant decline in model fit. Thus, a simple AE model provided an acceptable fit to the data for all four TPQ personality dimensions.

We were also able to apply a constrained AE model (i.e., same estimates for the two cohorts) without a significant decline in model fit from the unconstrained AE model (allowing different estimates for each age cohort) for RD, PS, and HA ($\chi^2_{(10)} = 5.99$, $p = .81$; $\chi^2_{(10)} = 7.77$, $p = .65$; $\chi^2_{(10)} = 15.1$, $p = .13$, respectively). For NS, a constrained AE model also provided an acceptable fit ($\chi^2_{(9)} = 13.79$, $p = .13$; $\Delta\chi^2_{(1)} = 0.21$, $p > .05$) if the total variance in the younger and older cohorts was allowed to dif-

fer (i.e., a scalar sex-limitation model was fit to the data).

Analysis 2

In analysis 2 we used age as a continuous variable and estimated expected phenotypic means, and genetic and environmental influences on individual differences, as a continuous function of age and age squared across the full age range from 50 to 96 years. Fig. 3 shows the number of twins at each age in our sample. Note that age information is limited at both extremes of the distribution. Thus, although our sample ranges in age from 50 to 96, there is insufficient data for individuals younger than 55 and older than 80 to generalize our findings beyond this age range.

Fig. 1b shows the observed means at each age for each of the TPQ dimensions. Fig. 2b shows the observed variances at each age across our age range. Note that except at the extreme end of the age distribution, where we have limited data, there is very little evidence for age/cohort differences in total variance. NS shows a significant decline in variance across the age range. PS shows increased variance, but only for the oldest age cohorts, where we have little data. The expected means—modeled as a continuous function of age and age squared—are shown in Fig. 4. Consistent with analysis 1, we see modest but significant linear declines in NS, RD, and PS. That is, linear trends were sufficient to describe the data. All quadratic effects on the means could be constrained to zero. No significant age differences were found for HA.

Fig. 5 shows standardized heritability (h^2) estimates across our age range, and Fig. 6 shows corresponding environmentality (e^2) estimates for the four scales. Estimates for the full model (including linear and quadratic age effects) are shown. None of the quadratic or linear effects were significant, however. For all four scales the 95% confidence intervals for the linear and quadratic parameter estimates included zero. In general, results from analysis 2 are consistent with analysis 1 findings and indicate modest age differences in means for three of the four TPQ dimensions. We found no evidence for substantial age differences in the proportion of genetic and environmental influences on individual differences on the higher-order TPQ dimensions across our age range. However, as noted earlier, there was a significant decline in total variance for NS.

Note that our analysis 1 results suggested that a "hybrid" ACE/ADE model (i.e., different models for

Table II. Variance Components of Model-Fitting Results for the TPQ Scales

Phenotype/group		a ²	c ²	d ²	e ²	χ ²	df	p	AIC
RD									
AE	Young	0.40			0.60	5.24	8	.732	−10.7
	Old	0.35			0.65				
ACE	Young	0.40	0		0.60	5.24	6	.513	−6.75
	Old	0.35	0		0.65				
ADE	Young	0.28		0.12	0.60	5.09	6	.532	−6.90
	Old	0.33		0.02	0.65				
Phenotype/group		a ²	c ²	d ²	e ²	χ ²	df	p	AIC
PS									
AE	Young	0.16			0.84	5.48	8	.705	−10.5
	Old	0.27			0.73				
ACE	Young	0.154	0.004		0.842	5.48	6	.483	−6.51
	Old	0.27	0		0.73				
ADE	Young	0.16		0	0.84	5.44	6	.489	−6.56
	Old	0.21		0.07	0.72				
Phenotype/group		a ²	c ²	d ²	e ²	χ ²	df	p	AIC
NS									
AE	Young	0.43			0.57	13.58	8	.093	−2.41
	Old	0.46			0.53				
ACE	Young	0.05	0.37		0.58	8.83	6	.183	−3.17
	Old	0.46	0		0.54				
ADE	Young	0.43		0	0.57	11.03	6	.087	−0.96
	Old	0		0.48	0.52				
Phenotype/group		a ²	c ²	d ²	e ²	χ ²	df	p	AIC
HA									
AE	Young	0.49			0.51	15.07	8	.058	−0.93
	Old	0.48			0.52				
ACE	Young	0.49	0		0.51	12.62	6	.049	0.62
	Old	0.23	0.23		0.54				
ADE	Young	0.08		0.41	0.51	14.17	6	.028	2.17
	Old	0.485		0	0.51				

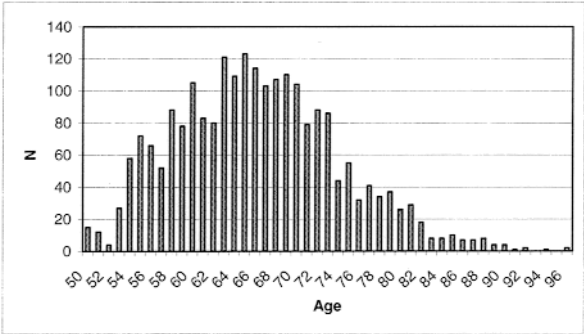


Fig. 3. Sample age information.

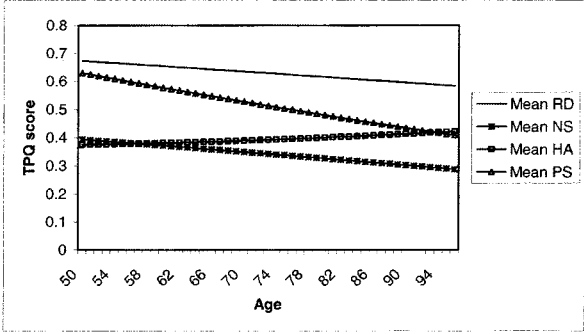


Fig. 4. Expected means as continuous function of age.

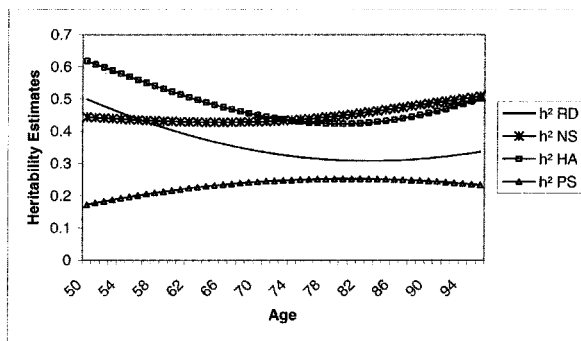


Fig. 5. Heritability estimates as a continuous function of age.

the younger and older cohorts) might provide a better fit to the NS and HA scales, and in fact those models did provide smaller, though not substantially smaller, omnibus χ^2 values (for NS $\chi^2_{(6)} = 6.28$, $p = .39$; for HA $\chi^2_{(6)} = 11.72$, $p = .07$). However, our analysis 2 results using continuous age did not show significant age differences for any of the genetic influences or environmental influences across our age range, suggesting that the pattern of results in analysis 1 that indicate a possible “hybrid” ACE/ADE model do not reflect real trends across the full range.

DISCUSSION

In this study we examined cross-sectional age differences in phenotypic means, the phenotypic variance-covariance structure, and the underlying genetic and environmental structure of four personality constructs from Cloninger’s personality system. Our sample consisted of same-sex female twins between the ages of 50 and 96, drawn from the American Association of Retired Persons (AARP) twin sample. We examined age differences by comparing younger (age 50–65) and

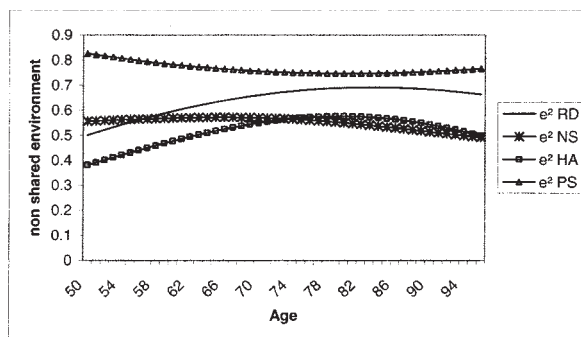


Fig. 6. Non-shared environment as a continuous function of age.

older (age 66+) cohorts (based on a median split of the sample), as well as estimating biometric model parameters as linear and quadratic functions of age. Typically, cross-sectional studies investigating age differences in personality have relied on group comparisons. The advantages of the analyses presented here are that they avoid arbitrary cutoffs to define age-groups and also allow for investigating nonlinear age/time of measurement trends that would be difficult to detect in group comparison analyses.

Consistent with the personality literature, we found moderate heritability estimates for HA, NS, and RD, ranging from 0.30 to 0.62 across our age range. Persistence showed the lowest heritability estimates, ranging from 0.16 to 0.27. It should be noted, however, that the Persistence scale is made up of a relatively small number of items compared with the other dimensions, and therefore the lower heritability for PS should be interpreted with some caution. More important, results from both our cohort and age-to-age comparisons suggested few cross-sectional age-related differences for the TPQ higher-order dimensions. We found relatively modest mean-level age/cohort differences across our older adult age range for three of the four TPQ personality dimensions. NS, RD, and PS showed significant declines of approximately half a standard deviation, while HA showed a slight, but non-significant, increase in mean level. With the exception of NS, there was no evidence for substantial age/cohort differences in phenotypic variances and covariances, nor in the genetic and environmental contributions to individual differences on the TPQ dimensions across our older adult age range. NS did show a 22% decline in total phenotypic variance, but the relative contribution of genetic and environmental influences on individual differences in NS did not show age/cohort differences across our older adult range.

Because there are no longitudinal or cross-sectional age-comparison studies utilizing the TPQ in later adulthood, direct comparisons of our data to other studies are not possible. However, there have been longitudinal studies of Extraversion (E) and Neuroticism (N) from the Eysenckian and Big Five personality models, and E and N have been shown to correlate moderately with Cloninger’s NS and HA. For example, in an earlier report on the current sample, Stallings *et al.* (1996) showed that the correlation between HA and N (as assessed by the EPQ-R; Eysenck, Eysenck, and Barrett, 1985) was .56, and the correlation between NS and E was .37. Heath, Cloninger, and Martin (1994) also showed that these dimensions tap overlapping con-

structs to some extent but are not alternative descriptions of the same dimensions of personality. Nonetheless, given the evidence for some overlap, we compared our findings for NS and HA to available longitudinal studies of E and N.

In a cross-cultural investigation of changes in the mean-level stability of personality (encompassing more than 10 different countries or cultures), McCrae *et al.* (1999, 2000) showed striking parallels of age differences and mean changes across samples. They found declines in mean levels for both E and N across all of the cultures examined. Such striking similarities in mean change across such diverse cultures led McCrae *et al.* to hypothesize that mean-level changes in personality are due to intrinsic maturational processes. A recent study by Loehlin and Martin (2001) also found a longitudinal decrease in mean scores for neuroticism in middle adulthood (mean ages for their longitudinal assessments were 47.5, 56, and 62). For extraversion they found a decrease in means from time 1 to time 2 but an increase from time 2 to time 3. Both E and N showed relatively stable variances across time. Loehlin and Martin's sample included both men and women, and their assessments at older ages would represent only the youngest cohort in our sample. A cross-sectional examination of the women only from Loehlin and Martin's study showed a decrease in means for E and N in three cohorts (with mean ages of 23, 37, and 61). In a cross-sequential study of E and N, Viken *et al.* (1994) found a general decrease in mean E scores associated with cross-sectional age in women from ages 18 to 23 (youngest cohort) to 54 to 59 (oldest cohort). They also found that women aged 48 to 53 showed a longitudinal decrease in E when measured 6 years later.

Mean-level stability of personality has been a matter of some dispute, however, and findings have not been consistent across longitudinal studies. In a longitudinal study of E, N, and Openness to Experience (O) from the Swedish Adoption/Twin Study of Aging (SATSA), Pederson and Reynolds (1998) found substantial mean-level stability across age, time, and cohort for all three personality traits. Our cross-sectional findings for NS showed declines consistent with those reported for E in some studies, while our results indicated no age differences across middle to late adulthood for HA.

The gerontology literature has also demonstrated increases in variability with increasing age, a pattern that is usually more pronounced in longitudinal studies than in cross-sectional ones (Sprott, 1988; Nelson and Dannefer, 1992). Our cross-sectional data indicated very little age-related difference in the variances for

HA, RD, and PS, while NS showed a decline in both mean level and total variance. Given this pattern, NS in older adult populations may be a good choice of phenotype for future analyses using the methods of Dolan and colleagues (Dolan, Molenaar, and Boomsma, 1989, 1991, 1992, 1994) that explicitly test the hypothesis that the structure of the means and covariances (within the context of the covariance analysis of twin data) can be modeled by the same genetic and environmental latent sources. Such analyses would be most powerful with longitudinal data. It is also possible that lower endorsement of NS items with increasing age may have resulted in a "shortened scale," which would be consistent with declines in both mean level and variance.

Some important limitations of our study should be noted. First, our analyses are cross-sectional in nature and thus confound age differences with time of measurement or birth cohort differences. Our sample was a volunteer sample of Caucasian women of generally middle-class socioeconomic status, and so our conclusions should be restricted to that population. Finally, although our sample ranges in age from 50 to 96 years, note the small numbers at both extremes of the age distribution. Thus, our "effective" age range is probably 55 to 80 years of age. Even here, the detection of more subtle age differences in genetic and environmental influences would likely require larger numbers of twins at each age.

Given these limitations, our findings suggest very little evidence for age/cohort differences in the contribution of genetic and environmental influences to individual differences on the higher-order TPQ dimensions, at least across middle adulthood. In future studies we plan to investigate age-related differences in the primary subscales that make up each of the TPQ higher-order dimensions. A narrower investigation of the subscales may shed more light on the unique genetic and environmental factors influencing the primary subscales. It is quite possible that important age-related differences in the primary subscales are masked when analyses are conducted at the level of the higher-order dimensions alone, and most studies to date have focused on higher-order personality dimensions. Investigating the TPQ subscales will also allow us to study whether the phenotypic factor structure changes with age. Finally, it is also quite possible that age per se is not a good index for detecting life course differences or changes in personality. Future analyses might profit from examining important life course milestones such as retirement, widowhood, or menopause as potential moderators of personality change in later adulthood.

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